fluid was rendered unfit to carry on the nutrition of the tissues, and that the arteries suffered early from this defective nutrition. The conclusions the author drew from his elaborate examination of the subject was, first, that cerebral hemorrhage, when associated with renal disease, is almost always found to be dependent upon rupture of one or more of the cerebral arteries, in consequence of certain morbid changes having taken place in their walls; secondly, that these changes in the walls of the vessels are induced by the altered state of the blood, the effect of advanced disease of the kidneys; and, lastly, that the enlargement of the heart is the immediate effect of the renal disease, conjointly, perhaps, with the alterations in the coats of the vessels. The paper concluded with some remarks upon the treatment of these cases, in which a tonic and stimulating plan, rather than a lowering one, was advocated, and two cases were given which seemed to justify it.—*Med. Times and Gaz.*, Feb. 14, 1863.

19. *Influence of Hypertrophy of the Heart and Diseases of the Cerebral Arteries in the Production of Apoplexy.*—Dr. A. Eulenburg has investigated this subject statistically in a prize thesis presented to the Medical Faculty at Berlin. In 42 cases of sanguineous cerebral apoplexy, abnormal conditions of the arteries at the base of the brain—hardening, calcareous deposits, and fatty degeneration—were found in 29: in 13 cases only were the large cerebral arteries free from disease. In 9 of the 42 cases there was hypertrophy of the left ventricle. Of the 29 cases in which disease of the cerebral arteries was present, there was also more or less extensive endocarditis in 17, alterations of the valves of the heart in 19, and hypertrophy of the left ventricle in 6 only. Dr. Eulenburg hence draws the conclusion that disease of the cerebral arteries is a much more frequent cause of apoplexy than cardiac hypertrophy.—*British Medical Journal*, Dec. 6, 1862, from *Virchow's Archiv*, and *Wiener Medicin. Wochenschr.*, Sept. 6, 1862.

20. *Embolism.*—An interesting case of embolia of the infundibulum of the right ventricle and pulmonary artery communicated to the Société Anatomique, of Paris, by M. Gouraud, has been made the subject of a report by M. Lancereaux. The following is M. Gouraud's *résumé*: "A healthy woman, aged 46 years, entered La Charité, for a fracture of the right leg, accompanied by considerable extravasation of blood. Scutter's apparatus was applied, and all went on well, the size of the limb lessening. After three weeks the apparatus was replaced by a starch bandage. On the following morning the patient was quite well, but, some hours later, violent palpitations of the heart occurred, the patient cried out, became livid, and was dead in a few minutes. On post-mortem examination, the right tibia presented two solutions of continuity, the fibula being fractured in only one place; there was an extravasation of blood infiltrating the whole thickness of the soft parts in this region. The veins of the right leg presented small coagula, which became more distinct and large in the femoral vein, the external and common iliac, and even in the lower part of the vena cava. The fibrinous coagulum was firm, elastic, of a deep red or rose colour, and was adherent at several points to the internal surface of the vessel. On the left side the limb and veins were healthy. From the lower part of the vena cava to the heart the blood was liquid. There existed in the infundibulum of the right ventricle and in the pulmonary artery a clot drawn out into the form of a leech, thirty-six centimetres in length, of a diameter much less than the vessel where it was found, of a rose or deep red colour, and not homogeneous. The lungs were engorged, but crepitant." M. Gouraud explains the obliteration of the passage by the arrest of the long clot, on arriving at a branch of the pulmonary artery, such as would not allow it to proceed further, and then by the ventricular contractions causing the other extremity to be folded back in the infundibulum, so as to lie opposite the sigmoid valves. It is necessary that we should abridge considerably M. Lancereaux's observations. The first question to which he applies himself is the cause of the coagulation which took place in the veins. He explains it thus: The blood coagulated at the seat of the fracture necessarily compressed the mouths of the ruptured vessels; but, at the same time, coagula would form at the extremities of these vessels, and mount up, as is the rule for

them to do, as high as the nearest valves. From the withdrawal of the *vis a tergo*, there would be stasis of the blood proceeding from the collateral veins, a new coagulum, commencing this time at the valves, and these latter coagula would lengthen gradually, and become, in their turn, the cause of new coagula, until the principal venous trunk becomes completely obstructed. In this view the cause is a local one, namely, the diminution of the current of blood, and the influence exercised by the fibrinous clot upon the blood which surrounds it. Admitting the sufficiency of this cause to produce venous coagulation, other causes may be added, such as diminution or loss of contractile power in the veins, tumours compressing them, and whatever retards the venous circulation. General causes would also operate, on the one hand, by lowering the force of the heart and the contractility of the vessels, and, on the other, by causing modifications in the blood itself such as are even now little understood. It is important to point out that, under the influence even of general causes, it is always where the circulation tends to be slow that coagulation commences.

The clot, which begins to be formed at the situation of a valve, presents a form and characters which must first be treated of. At one extremity it presents the mould of one or two of the valves; its other end is rounded or conical, and upon its length may be perceived the smooth and clean impressions of valves. One of its surfaces, that in contact with the wall, is strictly striated, yellowish or marbled; the other surface, free and bathed in the blood, is brownish and granular. The length varies from some millimetres to several centimetres; its bulk may become considerable, since it generally forms in the largest vessels, and is thus the most frequent cause of sudden deaths. Besides, by reason of its characters, it constitutes the most positive evidence of embolia of the pulmonary artery when it is met with in this entirely valveless vessel. Observation teaches us that, where there is but one clot, and the death has been sudden, it is always the trunk of the pulmonary artery or the infundibulum which is found obstructed. The blood in the heart is ordinarily black and fluid, as in death by asphyxia. I do not, for my own part, think that a single embolus, arrested in one of the divisions of the artery, can bring about this fatal accident. For the most part the embolic clots are multiple, and always, I say again, if death has been rapid, they are found either in the trunk of the pulmonary artery or in its principal branches. As respects the smallest clots, they are rarely found in divisions of the fifth order, but mostly in those of the third or fourth. In some special cases known as capillary emboli, very small clots have been found in the smallest branches. The form of the migratory clots is generally cylindrical, their extremities at one time regular, smooth, and conical; at another, rough and torn; at another, only one end is torn, while the other is polished and conical. It is in cases where both extremities are smooth and untorn that valvular impressions are found upon the body of the clot, and one or two moulds of valves at one extremity. The clots which are torn at their extremities are generally devoid of impression and moulds, but they are now and then channelled. When one extremity only is torn, the other is generally conical. The same difference which we have established in the characters of venous clots is, consequently, found in the clots of the pulmonary artery; and there exists between the venous coagula and those of the pulmonary artery such a resemblance, that we are compelled to admit that the clots have been transported from the veins into this artery.

Besides these characters, embolic clots differ from coagula formed just prior to or immediately after death in their elasticity, brownish or marbled colour, and the condition of the fibrin which is always in progress of retrogression. The clots formed at death are soft, œdematous, flattened, branched, and only close incompletely the containing vessel. The clots which, during life, form primarily in the branches of the pulmonary artery (autochthones), differ from emboli in their form and seat, and in the absence of the characters which have been described. In certain circumstances, however, they are readily confounded with embolic clots, namely, where fibrinous coagula have become added to the latter, but it is always easy, by means of a section and examination with the microscope, to recognize the central embolus. If the bulk of embolic clots is very variable, their length especially presents great variety: thus, whilst some

may only measure a few millimetres, others are several centimetres in length; such as I have seen produce sudden death, have been five centimetres long; that which M. Gouraud has described in his observation presented the extraordinary length of thirty-six centimetres. I am disposed to believe that some error has slipped into this measurement, especially seeing that the femoral and part of the iliac veins were filled with a fibrinous coagulum. Under these circumstances it is necessary to suppose that the embolic clot occupied primarily the greater extent of the vena cava, a hypothesis of little probability, since no symptom of such an obstruction was apparent during life. I am, consequently, driven to believe that some secondary coagulations have been comprised in the measurement. But be this as it may, it is certain that very long clots may be carried by the torrent of the circulation, and an important and peculiar character of them is, that they are curved and wound round, at one time in the trunk of the pulmonary artery, and in the infundibulum, as in M. Gouraud's case—at another, in one of the principal divisions of the pulmonary artery. But after a certain lapse of time these distinctive characters become wanting, and it is then very difficult to tell whether a coagulum, met with in the pulmonary artery, has been formed there, or has arrived there by migration. The only circumstance which we may thus be able to call up in favour of embolus, is the existence of a venous thrombus. The phenomenon which renders the embolus unrecognizable is important and really remarkable. The continued contact of the clot with the arterial wall determines a slight irritation, in virtue of which a blastema, exuded between the wall of the vessel, and the clot, soon becomes organized; by degrees, this substance extends on the circumference of the plug, and soon forms a sort of cupule, in which the latter is contained. At last it envelops it completely, and encysts it, so that, after a time, often not very long, the fibrinous coagulum of the pulmonary artery is found to be everywhere surrounded by a perfectly organized membrane. Within this membrane, microscopic examination discovers an amorphous substance, more or less granular, embryo-plastic nuclei, elongated cells, and, above all, fibres of connective tissue. In the midst of these elements we sometimes find capillaries, free granules, the *débris* of red globules, and amorphous and crystalline hæmatin. After describing further changes in the condition of these encysted clots, M. Lancereaux proceeds to the subject of the condition of the lungs in cases of pulmonary embolia. It is evident (he continues) that an embolus which closes up the trunk of the pulmonary artery, and gives rise to sudden death, cannot cause any important disorder in the pulmonary parenchyma. Supposing such alteration possible, time would be wanting. But it is different when a coagulum comes to be situated in an important division of the artery, closing its canal completely. In this respect, M. Lancereaux expresses his agreement with Virchow, who states that, however complete the obstruction, it produces no alteration in the parenchyma, and, above all, no gangrene of the lungs. At the most, Lancereaux has observed slight diminution of volume, anæmia, or some œdema; and he explains this, physiologically, by the fact, that the pulmonary artery is an organ engaged in hæmatosis, and that the nutrition of the lung is effected, not by this, but by the bronchial arteries. Still (he proceeds to say) pulmonary coagula are sometimes accompanied by a lesion of the parenchyma of the lungs, whether they be the cause of it or not. Pulmonary apoplexy is frequently conjoined with obstruction of the branches of the artery; but it is to be remembered, that this generally occurs in the course of affections of the heart, especially in fatty degeneration; and it is also to be observed, that, under these circumstances, the clot is always situated behind the apoplectic spot, has none of the characters of an embolic clot, and is evidently autochthonic—not the cause, but an effect, of the apoplexy. The same thing may happen in certain cases of tubercular disease, of pneumonia, or even of gangrene. It is, however, important to notice, that there are certain special conditions of the embolic clot which are capable of giving rise to two of the alterations just alluded to—namely, pneumonia and gangrene. These conditions pertain to a special state of alteration of the tissues, in the midst of which the thrombus has been formed: if the coagulation has taken place in the midst of a purulent or gangrenous focus, the coagulum, formed in part of fibrin, and in part of other elements, possesses qualities in virtue of which it may alter the

tissues with which it subsequently comes in contact; thus it is that metastatic abscesses often appear in the lungs of individuals, with suppurative thrombus of the cerebral sinuses, and in women suffering from metritis or suppurative phlebitis. Thus, too, gangrenous spots in the brain are found in persons who have primarily a gangrene of the lung; and gangrene of the lungs is met with frequently in paralytic individuals, in whom a sphacelus has formed over the region of the sacrum. Particles of fibrin or fragments of tissue, impregnated with pus or septic matters, become the points of origin of secondary foci, purulent or gangrenous, as the case may be.

Certain practical conclusions flowing from this fact related by M. Gourand deserve attention. We find here a condition which has already been mentioned in other cases, one of which is related by Klinger. In three different cases sudden death has followed shortly upon compression exercised by a bandage upon the limb, which is the subject of the thrombus. It was, as we know, formerly customary to apply a compressing bandage upon a limb affected with œdema, or even with phlebitis, as soon as the acute stage of the inflammation had ceased. The practice is far from being free from danger, and it must necessarily be proscribed. But, besides, when, consecutively to a traumatic condition, fracture, amputation, etc., we have reason to suspect the existence of a venous thrombus, it will surely be prudent in the surgeon to abstain as much as possible from strong compression, if he would not expose his patient to more or less serious accidents. It is especially some time after the commencement of the coagulation, when the fibrin begins to disintegrate, that we must avoid this practice. And, for the same reason, every kind of handling of the injured limb should be avoided, and the most complete state of rest maintained. Indeed, in a certain number of cases, a slightly exaggerated effort, as I have seen on two different occasions, may suffice to bring about the separation of the clot, and sudden death. Occasional causes of this kind are marked, in the greater number of cases of sudden death, by embolia, and it is thus pointed out how necessary it is to be cautious when we have to do with patients suffering from venous thrombus.—*Med. Times and Gaz.*, Feb. 14, 1863.

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21. *Conditions affecting the Constitution of Phthisical Persons when in Health.*—Dr. EDWARD SMITH, Assistant Physician to the Hospital for Consumption and Diseases of the Chest, has published (*Dublin Quarterly Journal of Medical Science*, February, 1863) a very interesting statistical inquiry into the prevalence of numerous conditions affecting the constitution in 10,000 phthisical persons when in health; intended to show the prevalence of a large number of conditions which are believed to modify the constitution, or which are evidences of modified constitutions in consumptive families. The investigation extended to a very large number of questions, and the results are given in separate tables.

The author gives the following summary of the leading truths which his extended investigation suggests:—

"The first question which arises is that of hereditary transmission, either in the sense of absolute transfer of the elements of the particular disease from the parent to the child, or the communication of a state of the system in which disease in general, and this disease in particular, may probably originate. There is a wide difference in these two ideas, and yet it cannot be doubted that they both exist in the minds of various professional men at this day. The former is the older one, and that which the increasing knowledge of our day has rendered less tenable than was formerly believed, since the idea of the transmission of the germs of disease *in utero* is now more strictly limited to such specific diseases as syphilis. Yet it must be admitted, that whilst the growing feeling of the day is in favour of a theory which only implies a defective constitution, there is an under current of belief that this assumes a specific direction in the production of this particular class of disease. Hence, whilst there is a clear distinction in the two theories in statement, there is far more oneness in belief. We will look at them in both aspects.

"Feebleness of the general health of the parents existed before the birth of the patient in one-fifth, and throughout life in one-third of all the cases. It is